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UNITED STATES DISTRICT COURT
DISTRICT OF UTAH, CENTRAL DIVISION

<p>LINDA SMITH, as Personal Representative on behalf of the Legal Heirs of RONNIE SMITH, Deceased, ,</p> <p>Plaintiff,</p> <p>vs.</p> <p>FORD MOTOR COMPANY, et al., ,</p> <p>Defendants.</p>	<p>MEMORANDUM IN SUPPORT OF FORD MOTOR COMPANY’S DAUBERT MOTION TO EXCLUDE EXPERT TESTIMONY OF SAMUEL HAMMAR, M.D.</p> <p>Case No. 2:08-cv-00630DB</p> <p>Judge Dee Benson</p>
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Defendant Ford Motor Company (“Ford” or “Defendant”) moves to preclude Plaintiff from introducing evidence or testimony through his expert, Dr. Samuel Hammar, that “every exposure” to asbestos-containing automotive friction products causes mesothelioma because the methodology behind this opinion does not meet the *Daubert* standard and lacks scientifically reliable support.

I. STATEMENT OF THE ISSUES AND SUMMARY OF THE ARGUMENT

This Court must determine whether Plaintiff’s medical expert witness, Dr. Samuel Hammar (“Dr. Hammar”), should be permitted to testify: (1) that Mr. Smith’s alleged exposure to asbestos-containing automotive products causes mesothelioma in humans, otherwise known as “general causation,” and (2) whether exposure to Ford’s products actually caused the specific injury to Mr. Smith, known as “specific causation.” Dr. Hammar’s so-called “every exposure” opinion, that is, that every single asbestos exposure from automotive friction products caused Mr. Smith’s alleged injuries, is unreliable and should not be admitted at trial because it lacks a reliable scientific or methodological foundation, and furthermore, is actually rebutted by numerous epidemiological studies.

Dr. Hammar can prove neither general causation—that exposures to brakes increases the risk of mesothelioma--nor can he prove specific causation. Dr. Hammar’s proffered specific causation opinion is based entirely on the notion that every exposure to asbestos contributes to the development of disease. This notion is an extrapolation from observations showing that those with high-dose exposures (e.g., miners, shipyard workers) have a greater risk of mesothelioma than unexposed individuals. In short, Dr. Hammar assumes that because high doses of asbestos (including the more potent amphibole forms of asbestos) have been found to cause mesothelioma, then low doses of less potent chrysotile must also cause mesothelioma.

This proposition is no different than reasoning that because taking 100 regular-dose aspirin in a day can kill humans, taking one low-dose aspirin in a day is also lethal. This leap of logic defies all recognized scientific principles, including the maxim of toxicology “the dose makes the poison.”¹ Because Dr. Hammar’s flawed methodology and unsupported conclusory opinion establish neither general nor specific causation, Plaintiff cannot satisfy her burden of proof.

This exact “every breath” opinion has been rejected by numerous courts. As is discussed further below, and in the affidavit of Dr. Mark Roberts, attached as Ex. 2, multiple epidemiological studies, and many recent case opinions, rebut the opinions of Plaintiff’s medical expert Dr. Hammar that “every exposure” to automotive friction products causes mesothelioma. *See, e.g., Betz v. Pneumo Abex*, 44 A.3d 27 (Pa. 2012); *Dixon v. Ford Motor Company*, September Term, 2011, No. 536 (Court of Special Appeals of Maryland, June 29, 2012) (holding that the “every exposure” theory provides no information that could assist the trier of fact in determining substantial factor causation). *See* attached as Ex’s 3 and 4.²

¹ It is a well-established scientific principle that “the dose makes the poison” and in 1538, Paracelsus observed “What is it that is not poison? All things are poisons and none that [is] not. Only the dose decides that a thing is not poisonous.” W.N. Aldridge, *The Biological Basis and Measurement of Thresholds*, Ann. Rev. Pharmacol. Toxicol. 1986. 26:39, 39, attached as Exhibit 1. The Federal Judicial Center has recognized that Paracelsus’ observation as a “central tenant of toxicology” and in its *Reference Guide on Toxicology* acknowledges that “the dose makes the poison”; this implies that all chemical agents are intrinsically hazardous—whether they cause harm is only a question of dose.” B. Goldstein & M.S. Henifin, *Reference Guide on Toxicology*, Reference Manual on Scientific Evidence, Federal Judicial Center, 401 (2d ed. 2000).

² *See also generally* Anderson et al., The “Any Exposure” Theory Round II – Court Review of Minimal Exposure Expert Testimony in Asbestos and Toxic Tort Litigation Since 2008, attached as Ex. 5

II. STATEMENT OF FACTS REGARDING THE NATURE OF PLAINTIFF'S ALLEGED ASBESTOS EXPOSURE

Plaintiff alleges that Ronnie Smith (“Mr. Smith”) was exposed to asbestos and asbestos-containing products at various locations over a 50-year period between 1958 and 2008. *See generally* Complaint & Exhibit A attached thereto, Ex. 6. More specifically, Plaintiff alleges that Mr. Smith contracted pleural mesothelioma in the course of working on his family farm, on family home construction, as a laborer for various construction companies or other entities, as a gas station attendant, as a soldier in the United States Army, and as an accountant. *See Id.* at ¶ 14. As a gas station attendant from 1966 to 1968, Mr. Smith performed only occasional brake replacements of less than once per month; he was never a full-time mechanic. R. Smith Trial Depo. pp. 77-78, relevant portions attached as Ex. 7; R. Smith Disc. Depo. pp.193-94, 199-200, relevant portions attached as Ex. 8. Despite Plaintiff’s contention that Mr. Smith was exposed to asbestos over a 50-year period, Plaintiff attributes Mr. Smith’s injury to work on a handful of Ford vehicles; he “guessed somewhere between four and six.. *See* Complaint & Exhibit A attached thereto, Ex. 6; R. Smith Trial Depo. At pp. 80-81, Ex. 7; R. Smith Disc. Depo, p. 200, Ex. 8. Beyond his work as a gas station attendant, Mr. Smith only performed one other brake job on a Ford vehicle in his lifetime, when he changed the brakes on a 1957 Ford vehicle in approximately 1962. *R. Smith Disc. Depo*, pp. 123, 260, Ex. 8. Initially, Mr. Smith testified that it was his “recollection that that was the first time the brake shoes had ever been changed on that thing.” He later testified that he was “absolutely certain” that the brakes he removed were original equipment, simply because the car had less than 50,000 miles on it. Yet, Mr. Smith admitted that, depending on how the car was driven, a person might not be able to get 50,000 miles on a set of brakes. *Id.* at pp. 123, 260. He didn’t have any personal knowledge of the prior

maintenance work performed on this 1957 Ford. *Id.* at p. 257. Mr. Smith purchased the replacement brakes for his 1957 Ford at Bradshaw Auto Parts in Richfield, Utah. Although he thinks he purchased Ford replacement brakes, “there could have been another brand....” In the end, he said, “I don’t know.” He did not remember purchasing these brakes at a Ford dealership. *Id.* pp. 260-62; R. Smith Trial Depo. p. 124-25, Ex. 7. Despite Mr. Smith’s broad claims, Bradshaw Auto Parts did not sell “any replacement brake components manufactured or supplied by Ford Motor Company” during the 1960s. Declaration of David C. Norton ¶¶ 9-10, Ex. 9. Also, contrary to Mr. Smith’s allegations, Ford never distributed or sold brake linings or brake pads under the name “Motorcraft” until 1999; Motorcraft brakes have never contained asbestos. *See* Affidavit of Mark K. Taylor, filed in *Holcomb v. Ford Motor Co.*, et al., Case No. A556412, Nev. 3d J. Dist. Ct., ¶ 4, Ex. 10.

Plaintiff alleges that in the course of Mr. Smith’s limited work on Ford motor vehicles, whether personal or in his capacity as a gas station attendant, Mr. Smith inhaled asbestos dust. Plaintiff has indicated that she intends to offer expert testimony that working with, or exposure to, such allegedly asbestos-containing products can cause mesothelioma in humans, and that Mr. Smith’s work with such products caused his respective injuries, or increased his risk of developing mesothelioma at a later date. The only offered proof of the requisite element of causation attempting to link Mr. Smith’s injuries to Ford products is the November 24, 2008 Expert Report of Samuel Hammar, M.D., F.C.C.P., F.A.C.P. The November 24, 2008 Expert Report of Samuel P. Hammar, M.D. (“Hammar Report”), Ex. 11.

Dr. Hammar, as Plaintiff’s sole causation expert, attempts to tie Mr. Smith’s exposures to Ford’s specific friction products through a general conclusory opinion that “[b]ased on Mr. Smith’s history of occupational and non-occupational exposure to asbestos, I conclude within a

reasonable degree of medical certainty that every occupational and bystander exposure to asbestos above background contributed to cause his right pleural epithelial mesothelioma.”

Hammar Report at 6, ¶ 8. Dr. Hammar makes no assessment of the background exposure of Mr. Smith. *See generally, id.*

III. ARGUMENT

A. Plaintiff’s Burden of Proof

1. **Plaintiff Must Establish that “But For” Mr. Smith’s Alleged Exposure to These Particular Asbestos-Containing Friction Products He Would Not Have Suffered His Alleged Injuries.**

To prevail against Ford in this action, Plaintiff must establish that Ford’s negligence caused Mr. Smith’s injuries.³ The Plaintiff’s burden is to “introduce evidence which affords a reasonable basis for the conclusion that it is more likely than not that the conduct of the defendant was a cause in fact of the result.” *Nelson By and Through Stuckman*, 919 P.2d at 574 (Utah 1996). The “mere possibility of such causation is not enough; and when the matter remains one of pure speculation or conjecture, or the probabilities are at best evenly balanced” the plaintiff’s claim fails. *Id.* Negligent conduct must be a substantial cause of the harm alleged. *Holmstrom v. C.R. England, Inc.*, 8 P.3d 281, 292 (Utah App. 2000).

In *Holmstrom*, the Court defined what must be proven to establish that the negligent conduct was a “substantial” cause of the harm. The Court explained:

The word “substantial” is used to denote the fact that the defendant’s conduct has such an effect in producing the harm as to lead reasonable men to regard it as a cause, using that word in the popular sense, in which there always lurks the idea of responsibility, rather than in the so-called “philosophic sense,” which includes every one of the great number of events without which any happening would not have occurred. Each of these events is a cause in the so-called “philosophic

³ The elements of a negligence action are: (1) a duty of reasonable care owed by the defendant to plaintiff; (2) a breach of that duty; (3) causation; and (4) damages. *Ladd v. Bowers Trucking, Inc.*, 264 P.3d 752, 755 -756 (Utah App. 2011).

sense,” yet the effect of many of them is so insignificant that no ordinary mind would think of them as causes.

8 P.3d 281 at 292. “[No] case has been found where the defendant’s act could be called a substantial factor when the event would have occurred without it.” *Id.* citing W. Page Keeton, *Prosser and Keeton on the Law of Torts* § 41, at 268 (5th ed. 1984). Regardless of the semantics, the Utah Model Jury Instruction Committee recognized that the term “substantial factor” is “essentially, the but for test of causation.” *See* Utah Model Jury Instruction Committee Advisory Notes, attached as Ex. 12, citing *Holmstrom v. C.R. England, Inc.*, 2000 UT App 239, 8 P.3d 281; *see also Doe v. Garcia*, 961 P.2d 1181, 1185 (Idaho 1998) (a “substantial factor” is “one that ‘in natural or probable sequence, produced the damage complained of’ or one ‘concurring with some other cause acting at the same time, which in combination with it, causes the damage’”); Dan B. Dobbs, *The Law of Torts* § 171, at 416 (2001) (criticizing the “substantial factor” test as a worthless “incantation”).

The analysis in Utah courts mirrors the Tenth Circuit Court of Appeals in *June v. Union Carbide Corp.*, 577 F.3d 1234 (10th Cir. 2009). In *June*, plaintiffs alleged that radiation from defendant’s facility caused their illness. Plaintiffs argued that to show causation, they only needed to show that the radiation exposure was a “substantial contributing cause of the injury.” In so doing, the plaintiffs introduced expert testimony that concluded that the radiation was a “substantial factor” in causing the plaintiffs’ illnesses, “intended to mean that the exposures were one of the variables that contributed to the observed health effect.” *Id.* at 1246. The expert defined “substantial” as an “amount that is not trivial.” *Id.* The expert, however, did not state that the radiation alone was the actual cause of the cancer. *Id.* The Tenth Circuit discussed the confusion created by the phrase “substantial contributing factor” and then decided, just as the Utah Model Jury Instructions Committee, that to be a substantial contributing factor the

negligent conduct must be either “(a) a ‘but-for’ cause of plaintiff’s injury or (b) a necessary component of a causal set that (probably) would have caused the injury in the absence of other causes.” *Id.* at 1245. The Tenth Circuit Court of Appeals affirmed the trial court’s exclusion of plaintiffs’ causation experts and entered summary judgment against plaintiffs. *Id.* at 1252.

The scientific methodology for reaching a conclusion about whether a substance is the cause a particular individual’s disease requires consideration of both general and specific causation. *See Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 881 (10th Cir. 2005); *Wright v. Willamette Ind., Inc.*, 91 F.3d 1105, 1106 (8th Cir. 1996). To show that an asbestos-containing product attributable to Ford was a “substantial contributing factor,” the Plaintiff must provide expert testimony sufficient to prove both general and specific causation. *See, e.g., McClain v. Metabolife Int’l, Inc.*, 401 F.3d 1233, 1237 (11th Cir. 2005); *Ladd v. Bowers Trucking, Inc.*, 264 P.3d at 755 -756 (where “the injury involves obscure medical factors which are beyond an ordinary lay person’s knowledge, necessitating speculation in making a finding, there must be expert testimony that the negligent act probably caused the injury.”) *see also Fox v. Brigham Young University*, 176 P.3d 446, 452 (Utah App. 2007) (same). Further, without general causation there can be no specific causation. *See, e.g., Rutigliano v. Valley Bus. Forms*, 929 F. Supp. 779, 783 (D.N.J. 1996) (*citing In re “Agent Orange” Prod. Liab. Litig.*, 611 F. Supp. 1223, 1250 (E.D.N.Y. 1985) for proposition that specific causation requires proof of general causation and exclusion of all other possible causes).

Under Utah’s causation standard, plaintiff must prove by a preponderance of evidence that “but-for” Mr. Smith’s alleged exposure to Ford brake products, he would not have developed mesothelioma. The *only* evidence that Plaintiff offers in this action to establish the necessary element of causation is the opinion of Dr. Hammar that “every exposure” contributed

to cause the disease . Because Dr. Hammar's causation opinions are improper, speculative extrapolation, as discussed below, Plaintiff's expert testimony does not meet the standard for causation.

2. To Establish General Causation, Plaintiff Must Show That Brakes Cause Mesothelioma

The first element that Plaintiff must prove is general causation, i.e., whether the substance at issue is capable of causing the particular disease. *Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 881 (10th Cir. 2005) (in a toxic tort case, a plaintiff must offer reliable evidence of general causation, which is whether a substance can cause a particular injury). In terms of statistical proof, to prove general causation, Plaintiff must establish not just that exposure to automotive products increased somewhat Mr. Smith's likelihood of contracting mesothelioma; Plaintiff must establish that the exposure caused a *statistically significant* increase in the likelihood of contracting the disease. *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 43 F.3d 1311, 1320 (9th Cir. 1995) (*Daubert II*). Therefore, to sustain their burden of proof against Ford, Plaintiff must first establish that exposure to automotive friction products increases the risk of mesothelioma to a reasonable degree of medical probability. *See Vanderwerf v. SmithKline Beecham Corp.*, 603 F.3d 842 (10th Cir. 2010).

3. To Establish Specific Causation, Plaintiffs Must Show That the Four to Six Brake Replacements on Ford Vehicles Caused Ronnie Smith's Injury

Specific causation is whether the substance actually caused the disease in the individual plaintiff. *Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 881 (10th Cir. 2005) (in a toxic tort case, a plaintiff must also offer reliable evidence of specific causation, which is whether a substance caused an individual's injury in a particular case). Assuming that Plaintiffs can establish general causation, they must also establish specific causation for their particular case.

The issue of “specific causation,” however, must be established through expert medical testimony that considers each Plaintiff’s unique exposure and medical history after the epidemiological foundation has been laid.

It is well established that a plaintiff in a toxic tort case must prove that he or she was exposed to and injured by a harmful substance manufactured by the defendant. . . . In order to carry this burden, a plaintiff must demonstrate the levels of exposure that are hazardous to human beings [*38] generally as well as the plaintiff’s actual level of exposure to the defendant’s toxic substance before he or she may recover.

Mitchell v. Gencorp, Inc., 165 F.3d 778, 781 (10th Cir. 1999). As with expert witnesses in other scientific fields, the opinion testimony of Plaintiff’s medical expert witnesses must satisfy the *Daubert* standard, and must be founded upon scientifically reliable methodologies. *See Elcock v. Kmart Corp.*, 233 F.3d 734, 745-46 (3d Cir. 2000). Plaintiffs’ medical experts cannot merely assume the existence of a “significant” exposure to automotive friction products. Rather, Plaintiffs’ proposed experts must be able to point to reliable methodology and data supporting the expert’s assumption that Plaintiffs experienced sufficient exposure comparable to data that supports their conclusion. *See United States v. Downing*, 753 F.2d 1224, 1237 (3d Cir. 1985).

“As courts have recognized, it is improper for an expert to presume that the plaintiff must have somehow been exposed to a high enough dose to exceed the threshold necessary to cause the illness, thereby justifying his initial diagnosis. This is circular reasoning.” *Mancuso v. Consolidated Edison Co.*, 967 F.Supp. 1437 (S.D.N.Y. 1997); *see also Cuevas v. E.I. DuPont DeNemours and Co.*, 956 F.Supp. 1306, 1312 (S.D. Miss. 1997) (holding that knowledge of the amount and duration of exposure are “essential element[s] to a valid toxicological opinion determining whether a chemical caused certain reactions”); *Wright v. Williamette Ind., Inc.*, 91 F.3d 1105, 1107 (8th Cir. 1996) (holding that “[a]t a minimum, . . . there must be evidence from

which the fact finder can conclude that the Plaintiff was exposed to levels of the agent that are known to cause the kind of harm that the plaintiff claims to suffer”).

Dr. Hammar makes no finding of general causation whatsoever, and therefore cannot make a finding of specific causation. *Rutigliano*, 929 F. Supp. at 783 (D.N.J. 1996). Even as to specific causation, Dr. Hammar makes no analysis as to the dose experienced by Mr. Smith, but merely assumes that “every occupational and bystander exposure to asbestos above background” contributed to Mr. Smith’s mesothelioma. Ex. 11, ¶ 8. Dr. Hammar’s opinion provides no proper basis nor reliable methodology for evaluating Mr. Smith’s exposure, dose or whether such a dose is associated with disease.

B. Standard for Daubert

Rule 702 of the Federal Rules of Evidence specifies that if an expert’s opinion “will assist the trier of fact to understand the evidence or to determine a fact in issue,” that expert may testify “if (1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case.” This rule embodies three distinct substantive restrictions on the admission of expert testimony. Expert testimony must: (1) be qualified; (2) be based on reliable and scientifically valid methodology; and (3) “fit” the facts of the case. *See Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579, 589 (1993); *Oddi v. Ford Motor Co.*, 234 F.3d 136, 144 (3d Cir. 2000). The proponent of the expert testimony bears the burden of establishing admissibility by a preponderance of the evidence. *Ralston v. Smith & Nephew Richards, Inc.*, 275 F.3d 965, 970 n.4 (10th Cir. 2001).

In *Daubert*, the Supreme Court established that federal district courts retain a “gatekeeping role” over expert testimony and must “make certain that an expert, whether basing testimony on professional studies or personal experience, employs in the courtroom the same

level of intellectual rigor that characterizes the practice of an expert in the relevant field.”

Kumho Tire Co. v. Carmichael, 526 U.S. 137, 152 (1999). Consistent with this gate-keeping function, the *Daubert* Court directed trial courts to ensure that evidence presented by an expert witness is relevant, reliable and helpful to the fact-finder’s evaluation of the evidence. *Daubert*, 509 U.S. at 597. Helpfulness to the trier of fact is the “ultimate touchstone” for admissibility of expert testimony. *In re Paoli Railroad Yard PCB Litigation*, 916 F.2d 829, 857 (3d Cir. 1990) (“*Paoli I*”).

The party offering expert opinion has the burden of demonstrating that it is admissible. *See id.* at 744. Specifically, “the burden of establishing admissibility by a preponderance of the evidence . . . is on the proponent.” *Padillas v. Stork-Gamco, Inc.*, 186 F.3d 412, 418 (3d Cir. 1999). The proponent of the expert testimony must prove “that their [expert’s] opinions are reliable” and are based on “good grounds.” *In re Paoli R.R. Yard PCB Litigation*, 35 F.3d 717, 744 (3d Cir. 1994) (“*Paoli II*”); *Kannankeril v. Terminix Int’l, Inc.*, 128 F.3d 802, 807 (3d Cir. 1997).

1. Reliable Methodology

An expert’s opinions must be based upon valid reasoning and a reliable methodology. *Elcock v. Kmart Corp.*, 233 F.3d 734, 745-46 (3d Cir. 2000); *Kannankeril v. Terminix Int’l*, 128 F.3d 802, 806 (3d Cir. 1997). The focus “must be solely on principles and methodology, not on the conclusions that they generate.” *Oddi*, 234 F.3d at 145. As a result, an expert cannot rely upon mere subjective belief and speculation. *Daubert*, 509 U.S. at 590. Unsupported speculation or conclusory, subjective beliefs do not suffice to meet the requisite degree of “reliability” required. Simply put, this Court’s gate-keeping function requires more than “taking the expert’s word for it.” Fed. R. Evid. 702 Advisory Committee Notes.

Furthermore, the expert must explain his methodology. A district court need not admit opinion evidence where there is “too great of an analytical gap between the data and the opinion offered.” *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997). When an expert fails to clearly explain his method “in rigorous detail” and makes it nearly impossible to duplicate the expert’s subjective methods, there exists no testable hypothesis for which there are controlling standards. *See, e.g., Paoli II*, 35 F.3d at 742. Where an expert’s methodology is “arbitrary and not based on any recognized methodology,” it is “too speculative to pass muster under Rule 702.” *United States v. Lightman*, No. 92-4710, 1999 U.S. Dist. LEXIS 21646, at *7-8 (D.N.J. 1999) (excluding expert testimony where the expert failed to demonstrate support for the assumption that distribution of contaminants by a factor of two would also increase costs by a factor of two).

2. Fit

Rule 702 also requires that the expert’s testimony “fit” the case by assisting the trier of fact in resolving disputed factual issues. *See Oddi*, 234 F.3d at 145; *Paoli II*, 35 F.3d at 741-43. Even if an expert is sufficiently qualified and has “good grounds” for an opinion, there must be a “connection between the scientific research or test result to be presented, and the particular disputed factual issues in the case.” *United States v. Downing*, 753 F.2d 1224, 1237 (3d Cir. 1985). Where there is no logical connection between the facts in the case and the expert’s “expertise” or opinion, the proposed testimony does not “fit.” *See Meinhardt v. Unisys Corp. (In re Unisys Sav. Plan Litig.)*, 173 F.3d 145, 155-56 (3d Cir. 1999). “Nothing in either *Daubert* or the Federal Rules of Evidence requires a district court to admit opinion evidence which is connected to existing data only by the *ipse dixit* of the expert.” *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997); *Elcock*, 233 F.3d at 749.

C. **Dr. Hammar’s Conclusory “Every Exposure” Causation Opinion is Inadmissible Because He Employs a Flawed Methodology and His Opinion Lacks Scientifically Reliable Support.**

The conclusory opinion of Dr. Hammar, intended to establish both specific and general causation, is unsupported and based on fundamentally flawed and unreliable methodology. While Dr. Hammar's opinion makes an attempt to show specific causation of Mr. Smith's injuries, Dr. Hammar does not, and cannot, quantify a dose or exposure to Ford brakes by Mr. Smith, let alone a dose which increased his risk of mesothelioma. His baseless and speculative opinion cannot show that Mr. Smith's dose of asbestos from Ford friction products is associated with Mr. Smith's disease. Dr. Hammar's assumption, without analysis, that automotive friction products cause mesothelioma is unsupported by the science in the relevant field, and in fact is contradicted, and therefore his methodology is unreliable and his conclusion is inadmissible.

1. Dr. Hammar's Specific Causation Opinion is Unsupported and Based on Flawed Methodology Because Smith Was Not Exposed to Significant Quantities of Chrysotile Asbestos Fibers from Automotive Friction Products

Dr. Hammar's methodology, or at least what can be gleaned from his conclusory opinion, resorts to a chain: from chrysotile is carcinogenic, to brakes containing chrysotile, to brakes releasing chrysotile, to people breathing chrysotile fibers, and finally to people developing disease. However, this "methodology" fails to demonstrate a causal relationship between exposure to friction products and mesothelioma. At best, Dr. Hammar's steps lead to the conclusion that some people exposed to some level of chrysotile get mesothelioma, and that some people who worked with brakes have developed mesothelioma. But they also equally lead to the conclusion that some people exposed to chrysotile from brakes or from other sources do not get mesothelioma. What is evident is that Dr. Hammar's assumptions do not support the conclusion that a particular individual, in this case, Mr. Smith, developed mesothelioma that was more likely than not caused by the exposures to friction products (as opposed to any other commercial asbestos exposures, background asbestos exposures, or from some unknown causes).

Instead, Dr. Hammar improperly extrapolates from high dose-high potency asbestos to low dose, low potency asbestos exposures, and treats those exposures as if they have the same disease causing potential. Testimony by an expert does not become scientific knowledge just because it is presented by a scientist; nor can an expert's self-serving assertion that his conclusions were derived by the scientific method be deemed conclusive. *Daubert v. Merrell Dow Pharms.*, 43 F.3d 1311, 1315-16 (9th Cir. 1995) (on remand).

In federal courts, expert opinions based on extrapolation illustrate the nature of the “strong basis” and “strong logical inference” that must be shown for an extrapolated opinion to be considered admissible. For example, in *Rider v. Sandoz Pharms.*, 295 F.3d 1194 (11th Cir. 2002), the plaintiffs asserted that Parlodel caused them to experience hemorrhagic strokes. The Eleventh Circuit concluded that the district court did not err in finding that extrapolation from animal studies to humans was unreliable under *Daubert*, given that plaintiffs did not “offer some rationale for the suggestion that the vascular structures of humans and animals are sufficiently similar in this context to conclude that bromocriptine's effects on animals may be extrapolated to humans.” *Id.* at 1202. *See also Hollander v. Sandoz Pharms. Corp.*, 289 F.3d 1193, 1209 (10th Cir. 2002) (noting a lack of similarity between animal studies and human studies, including dose and route administration); *Tyler v. Sterling Drug., Inc.*, 19 F.Supp.2d 1239, 1244 (N.D. Okla. 1998) (““Test results on animals not necessarily reliable evidence of the same reaction in humans.”).

In *Schudel v. Gen. Elec. Co.*, 120 F.3d 991 (9th Cir. 1997), the issue at question was whether the plaintiff suffered from solvent-induced toxic encephalopathy caused by her exposure to two solvents. Her expert based his conclusions on extrapolation from studies that involved different solvents and different types of exposures than plaintiff sustained (e.g., the studies

examined short-term exposure at very high concentrations and plaintiff sustained short-term moderate level exposures). *Schudel*, 120 F.3d at 997. The Ninth Circuit concluded that the expert's testimony did not satisfy *Daubert* because the expert "did not establish it was scientifically acceptable to draw general conclusions about the neurotoxicity of TCA and Perc from studies of other chemicals; indeed, the testimony indicated small differences in molecular structure often have significant consequences." *Id.*; see also *Moore v. Ashland Chem., Inc.*, 151 F.3d 269, 279 (5th Cir. 1998) ("Several post-*Daubert* cases have cautioned about leaping from an accepted scientific premise to an unsupported one . . . To support a conclusion based on such reasoning, the extrapolation or leap from one chemical to another must be reasonable and scientifically valid.")

Other courts have also rejected extrapolation as not generally acceptable when it involves drawing conclusions regarding the biological effects of low doses of a substance from information regarding effects at high doses (i.e., extrapolating "down" a dose-response curve). In a pharmaceutical products liability case, the Northern District of California rejected an expert's downward extrapolation, reasoning:

The justification for extrapolation drug effects between biologically similar demographic groups, however, does not logically extend to the argument that all doses of a compound are harmful; accordingly, plaintiff's experts could not cite to a single piece of evidence that suggests that their experts' extrapolation is scientifically valid. To the contrary, with nearly all compounds there is usually a threshold that must be met before there is any harm; for example, even water can be harmful if consumed at certain amounts even though there is no harm at smaller amounts.

In re: Bextra & Celebrex Mktg. Sales Practices & Prod. Liab. Litig., 524 F. Supp. 2d 1166, 1180 (N.D. Cal. 2007). The Southern District of Florida rejected an expert's attempt to extrapolate from studies showing teratogenic effects of high doses of Vitamin A to low doses from Retin-A, which contains a derivative of Vitamin A. *Chikovsky v. Ortho Pharm. Corp.*, 832 F. Supp. 341,

345-46 (S.D. Fla. 1993). In rejecting downward extrapolation as an accepted methodology, the court expressed concerns that the expert “performed no comparisons between the dose of Vitamin A in the study and that found in Retin-A.” *Id.* See also *Baker v. Chevron USA, Inc.*, 680 F. Supp. 2d 865, 878 n.9 (S.D. Ohio 2010) (“The ‘one-hit’ or ‘no threshold’ theory of causation in which exposure to one molecule of a cancer-causing agent has some finite possibility of causing a genetic mutation leading to cancer is not a reliable theory for causation under Daubert standards.”); *Sutera v. Perrier Grp. of Am., Inc.*, 986 F. Supp. 655, 666 (D. Mass. 1997) (“There is no scientific evidence that the linear no-safe threshold analysis is an acceptable scientific technique used by experts in determining causation in an individual instance”).

Extrapolation is particularly problematic when it is used to produce conclusions that are inconsistent with extant studies. This was made clear in *Richardson v. Richardson-Merrell, Inc.*, 857 F.2d 823, 832 (D.C. Cir. 1988), which held that “courts should be very reluctant to alter a jury’s verdict when the causation issue is novel and ‘stand[s] at the frontier of current medical and epidemiological inquiry.’”

In each of these cases, extrapolation was rejected because the information used as the basis for the opinion was not sufficiently connected to the object of that position. Furthermore, the mere fact that some types of exposures to asbestos, in certain doses, cause mesothelioma is insufficient to support Dr. Hammar’s opinion that all exposures cause mesothelioma. Simply because a large exposure to a substance is associated with disease does not mean that much lower or different exposures cause disease. See, e.g., *Newkirk v. Conagra Foods, Inc.*, 727 F. Supp. 2d 1006, 1019 (E.D. Wash. 2010) (rejecting an expert’s causation opinions based upon a downward extrapolation from a high exposure to a significantly lower exposure in a “popcorn lung” disease case as unreliable because “he provides no basis to extrapolate . . . that slurry

vapors are the same whether inhaled over a tank at a popcorn plant or from a bag of microwave popcorn”).

Mesothelioma is a dose-responsive disease. Dr. Hammar has admitted, in other cases, that concentration and exposure are factors involved with an individual’s development of mesothelioma.

Q -- but what are the factors involved, then, with respect to an individual's development of mesothelioma?

A Well, I think probably one of the first factors would be genetic susceptibility or individual susceptibility. The next would be always is going to be a concentration issue, how much they were exposed to with the ideas that the more you are exposed to of asbestos, the greater your risk is of developing one of these asbestos-related diseases.

In re Personal Injury Asbestos Litigation, No. 24X09000164, Circuit Court for Baltimore City, March 22, 2011 Trial Testimony of Dr. Hammar, 1475:15-1476:4, attached as Ex. 13

Dr. Hammar’s opinion is based on none of this type of information. He makes no attempt to explain the process by which a single exposure to chrysotile contributes to mesothelioma’s development in the human body, let alone the particular exposures, dose, and body of Mr. Smith. Instead, Dr. Hammar’s every exposure opinion basically states that “where someone has exposure to asbestos, and they are diagnosed with mesothelioma, exposure plus diagnosis equals causation.” *In re: Asbestos Products Liability Litigation*, MDL No. 875, Case No. 2:05-90, Southern District of Georgia, September 1, 2010 Deposition of Samuel Hammar, M.D., 133:3-6, Ex. 14.

The idea that any exposure to asbestos, no matter how small or to what type of asbestos, leads to an increase in mesothelioma risk is unsupported, and in fact contradicted, by a great deal of evidence and depends in large part on extrapolations of dose response curves from high exposure conditions over several orders of magnitude to much lower exposure conditions, even

down to the levels of ambient asbestos encountered by the general population. A. Churg & F. Green, *Pathology of Occupational Lung Disease* 341 (2d ed. 1998).

2. Courts Reject the “Every Exposure” Opinion in Asbestos Cases

Recent cases throughout the country have recently rejected the “every breath” opinion that Dr. Hammar purports to submit in this case. *See, e.g., Betz v. Pneumo Abex*, 44 A.3d 27 (Pa. 2012); *Dixon v. Ford Motor Company*, September Term, 2011, No. 536 (Court of Special Appeals of Maryland, June 29, 2012) (holding that the “every exposure” theory provided no information that could assist the trier of fact in determining substantial factor causation), Ex. 4. Further, Dr. Hammar was precluded from testifying to the “every breath” opinion in *Free v. Ametec, Inc., et al.*, No. 07-2-04091-9SEA (Superior Court of the State of Washington in and for the County of King), Ex. 15.

In *Betz*, involving a career mechanic who claimed his mesothelioma was caused by brake exposures, the Pennsylvania Supreme Court unanimously held that plaintiff’s expert opinion that each breath of even one single asbestos from a defendant’s product was a substantial factor in the development of plaintiff’s asbestos-related disease is not supported by an accepted scientific methodology, and accordingly, did not meet the standard for admissibility of scientific opinion. *Betz*, 44 A.3d at 90. The Pennsylvania Supreme Court found that “[plaintiff’s expert’s] any-exposure opinion is in irreconcilable conflict with itself. Simply put, one cannot simultaneously maintain that a single fiber among millions is substantively causative, while also conceding that a disease is dose responsive.” *Id.* at 56.

Like the opinion the Pennsylvania Supreme Court held invalid in *Betz*, Dr. Hammar offers his specific causation opinion based solely on Mr. Smith’s mesothelioma diagnosis and a showing that he has breathed in at least one asbestos fiber. Dr. Hammar does not distinguish among Mr. Smith’s alleged lifetime exposures and presents no evidence regarding dose. Rather,

Dr. Hammar simply lumps all exposures together, opining that “[b]ased on Mr. Smith’s history of occupational and non-occupational exposure to asbestos, I conclude within a reasonable degree of medical certainty that every occupational and bystander exposure to asbestos above background contributed to cause his right pleural epithelial mesothelioma.” Hammar Report at 6, ¶ 8, Ex. .

Further, Dr. Hammer was precluded from testifying in the *Free* case after motions and a *Frye* hearing were held before the Honorable Suzanne Barnett of the Superior Court of the State of Washington, King County. The court held that “the assumption that every exposure to asbestos over a life’s work history, even every exposure greater than 0.1 fibers per cc year contributes to the disease, is not a scientifically proved proposition that is generally accepted in the field of epidemiology, pulmonary pathology, or any other field relevant to this case.” *Free v. Ametec, Inc., et al.*, Ex. 15.

Other courts have found testimony similar to that offered Dr. Hammar inadmissible under *Daubert*. For example, the Georgia Superior Court rejected the testimony of a Plaintiff’s expert pathologist who similarly opined that each and every exposure to asbestos causes disease. *Butler v. Union Carbide Corp.*, CA No. 2008CA114 (Ga. Sup. Ct. Morgan Cty. July 29, 2010), slip op. attached as Ex. 16. In *Butler*, the court deemed the expert’s opinion unreliable, because the expert’s each and every exposure theory had not been tested and it was impracticable to test this “theory.” *Id.* at 8. In rejecting the pathologist’s opinions, the court recognized that the “any exposure” theory failed to account for the large portion of the population that is exposed to asbestos and do not develop mesothelioma. *Id.* at 11. The court concluded that the “each and every exposure” theory was, at most, “scientifically-grounded speculation,” *id.* at 15, and

Daubert prohibits experts from speculating “about what they concede is not known by use of the scientific method.” *Id.* at 12.

3. Dr. Hammar’s General Causation Assumption is Unsupported Because Scientific Studies Show Brakes Do Not Cause Mesothelioma

Dr. Hammar simply assumes that exposure to automotive friction products causes mesothelioma. However, as discussed below, this assumption is unsupported, and in fact contradicted, by the science in the relevant field; therefore Dr. Hammar’s methodology is flawed and unreliable. Dr. Hammar does not, and cannot show that a dose of chrysotile from brake dust is associated with mesothelioma because he does not, and cannot, show that brake dust has toxicologic potential to cause disease.

a. The Chrysotile Asbestos in Automotive Brakes is Thermally Transformed Into Harmless Forsterite During the Braking Process

The asbestos in automotive friction products is unlike that in other types of products. There are two types of asbestos, amphibole and serpentine. Friction products contain chrysotile, a form of serpentine asbestos which is the least potent form of asbestos. Some estimates suggest that chrysotile is as much as 500 times less potent than crocidolite, a form of amphibole asbestos, and 100 times less potent than amosite. Affidavit of Dr. Mark Roberts, Ex. 2. Chrysotile also has a much shorter half-life than amphiboles, is more soluble, and breaks down more rapidly in the body. *Id.*

Even further, the asbestos contained in brakes is encapsulated in resins. When the brakes require changing, very little of the asbestos has survived the braking process. Rather, the chemical composition of chrysotile in brake linings changes, turning instead to an amorphous product known as forsterite, which is not considered carcinogenic. Brake dust, the material with which auto mechanics generally come into contact, is more than 99% forsterite, and less than 1%

asbestos in a non-fibrous form. *See e.g.*, Jeremiah R. Lynch, *Brake Lining Decomposition Products*, J. Air Pollution Control Ass'n, Dec. 1968, at 824, 826 (finding that more than 99% of brake linings were transformed to non-fibrous material as a result of the intense heat generated by the braking process), Ex. 17; Ronald L. Williams & Jean L. Muhlbaier, *Asbestos Brake Emissions*, *Envtl. Res.*, Oct. 1982, at 70-82, Ex. 18.

In general, auto mechanic exposure to chrysotile is considered to be low (in terms of level, frequency, and duration), and has been shown to be below regulatory limits. Brake wear dust is not “asbestos-containing” as defined by the EPA and the United States Department of Labor. Under those agencies’ definitions, a product is not considered to be an asbestos-containing product or material unless it contains more than 1% asbestos by weight or volume. 40 CFR § 763.163; 29 CFR § 1926.1001(b). Thus, brake dust – which consists of more than 99% non-hazardous forsterite – is not considered an asbestos-containing product under Federal guidelines.

b. Epidemiological Studies Show No Increased Risk of Mesothelioma in Automobile Mechanics

Epidemiological studies have consistently reported findings that auto mechanics do not have an increased risk of mesothelioma compared to the general population, therefore, Dr. Hammar’s conclusion is not only unsupported the relevant science, but also contradicted by it. *See, e.g.*, M. Goodman, et al., *Mesothelioma and Lung Cancer Among Motor Vehicle Mechanics: a Meta-Analysis*, 48 Ann. Occup. Hyg. 309, 322 (2004), Ex. 19-12.⁴ In fact, in his

⁴ At least 21 epidemiological studies have reported findings showing no increased risk of developing disease for mechanics, Affidavit of Dr. Mark Roberts, Ex. 2 at ¶ 18, including 16 peer-reviewed studies published in scientific journals. *See, e.g.*, (1) A.D. McDonald & J.C. McDonald, Malignant mesothelioma in North America, *Cancer* 46(7):1650-1656 (1980); (2) M.J. Teta, H.C. Lewinsohn, et al., Mesothelioma in Connecticut, 1957–1977, Occupational and geographic associations, *J. Occup. Med.* 25(10):749-756 (1983); (3) R. Spirtas, R. Keehn, et al., Mesothelioma risk related to occupational or other asbestos exposure: Preliminary results from a

deposition in this case, Dr. Hammar concedes that the latest studies have shown that there is no increased incidence of mesothelioma in automobile mechanics:

Q Have you been provided a summary or told of its conclusions with regard to the risk of mesothelioma involving asbestos friction products, repair of motor vehicles, et cetera?

A. It's my understanding that the latest studies there have shown no increased incidence of mesothelioma in automobile mechanics.

November 30, 2010 Deposition of Samuel P. Hammar, M.D., at 14:22-15:3, Ex. 20.

case control study, *Am. J. Epidemiol.* 122(3):518 (1985); (4) B. Järholm & J. Brisman, Asbestos associated with tumors in car mechanics, *Br. J. Ind. Med.* 45(9):645-646 (1988); (5) P. Gustavsson, et al., Lung cancer and exposure to diesel exhaust among bus garage workers, *Scand. J. Work Environ. Health* 16:348-354 (1990); (6) H.J. Weitowitz & K. Rödelberger, Mesothelioma among car mechanics?, *Ann. Occup. Hyg.* 38(4):635-638 (1994); (7) R. Spirtas, E.F. Heineman, et al., Malignant mesothelioma: Attributable risk of asbestos exposure, *Occup. Environ. Med.* 51:804-811 (1994); (8) D. Coggon, H. Inskip, et al., Differences in occupational mortality from pleural cancer, peritoneal cancer, and asbestosis, *Occup. Environ. Med.* 52:775-777 (1995); (9) K. Teschke, M.S. Morgan, et al., Mesothelioma surveillance to locate sources of exposure to asbestos, *Can. J. Public Health* 88(3):163-168 (1997); (10) J.T. Hodgson, J. Peto, et al., Mesothelioma mortality in Britain: Patterns by birth cohort and occupation, *Ann. Occup. Hyg.* 41:129-133 (1997); (11) A. Agudo, C.A. Gonzalez, et al., Occupation and risk of malignant pleural mesothelioma: A case-control study in Spain, *Am. J. Ind. Med.* 37:159-168 (2000); (12) M. Goodman, et al., Mesothelioma and lung cancer among motor vehicle mechanics: a meta-analysis, *Ann. Occup. Hygiene*, 48: 309-326 (2004); (13) P.A. Hessel, et al., Mesothelioma Among Brake Mechanics: An Expanded Analysis of a Case-Control Study, *Risk Anal.*, 24(3):547-552 (2004); (14) Peto, et al., Occupational, Domestic and Environmental Mesothelioma Risks in Britain: A Case-Control Study, *UK HEALTH AND SAFETY EXECUTIVE* (2009); (15) P. Rolland, et al., Pleural mesothelioma: Professions and occupational areas at risk among humans. Based on data from the French National Mesothelioma Monitoring Program (PNSM). Institut de Veille Sanitaire. Departement sante travail (2005); (16) C. Rake, C. Gilham, J. Hatch, A. Darnton, J. Hodgson, and J. Peto, Occupational, domestic and environmental mesothelioma risks in the British population: a case-control study, *British Journal of Cancer* (2009) 100, 1175 – 1183 (2009); and (17) P. Rolland, et al., Occupations and Industries in France at High Risk for Pleural Mesothelioma: A Population-Based Case-Control Study (1998–2002), *Am. J. Ind. Med.* (2010), 53: 1207-1219, collectively attached as Exhibits 19-1 through 19-17.

As a general matter, epidemiological studies are considered the best evidence of a causal relationship between a toxin and a disease. Because epidemiology is “the medical science devoted to determining the cause of disease in human beings,” *see e.g., Siharath v Sandoz Pharmaceuticals Corp.*, 131 F.Supp.2d 1347, 1356 (N.D. Ga. 2001), courts have long recognized that epidemiological studies provide the best, most reliable evidence, of whether exposure to a particular substance is capable of causing a particular disease. *See Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 882 (10th Cir. 2005) (“We agree with the district court that epidemiology is the best evidence of general causation in a toxic tort”); *see also See, e.g., Rider v. Sandoz Pharms. Corp.*, 295 F.3d 1194, 1198 (11th Cir. 2002) (“Epidemiology, a field that concerns itself with finding the causal nexus between external factors and disease, is generally considered to be the best evidence of causation in toxic tort actions.”); *Brock v Merrell Dow Pharmaceuticals, Inc.*, 874 F.2d 307, 315 (5th Cir. 1989) (concluding that “[u]ndoubtedly, the most useful and conclusive type of evidence in a [toxic tort case] is epidemiological studies”); *Siharath, supra*, 131 F.Supp.2d at 1356 (“epidemiological studies provide ‘*the primary generally accepted methodology* for demonstrating a causal relationship between a chemical compound and a set of symptoms or disease.’”) (emphasis added); M. Green, Expert Witnesses and Sufficiency of Evidence in Toxic Substances Litigation: The Legacy of Agent Orange and Bendectin Litigation, 86 Nw. U.L. Rev. 643, 646 (1992) (“The most desirable evidence is epidemiologic, because it can best be generalized to support inferences about the effect of an agent in causing disease in humans.”).

The Fifth Circuit Court of Appeals has succinctly stated that “speculation unconfirmed by epidemiologic proof cannot form the basis for causation in a court of law.” *Brock, supra*, 874 F.2d at 315; *see also Siharath, supra*, 131 F.Supp.2d at 1358 (explaining that “[t]he absence of

epidemiological support raises the question of whether the causation opinions of Plaintiffs' experts are merely speculative and not based on scientific knowledge"); *Bell v Swift Adhesives, Inc.*, 804 F.Supp. 1577, 1579 (S.D. Ga. 1992) (holding that animal studies are not capable of proving causation in human beings absent confirmatory epidemiological data).

In *General Electric Co v Joiner*, 522 U.S. 136 (1997), the United States Supreme Court considered the sufficiency of expert testimony purporting to link Mr. Joiner's exposure to PCBs and his lung cancer. The Supreme Court held that the district court properly rejected expert testimony based on animal studies rather than epidemiological research. In addition, the court considered four epidemiological studies relied upon by plaintiff's experts, and determined that none of the studies supported a causal link. In two of the epidemiological studies, the *authors refused to recognize a connection* between PCB exposure and lung cancer. The third study made no mention of PCBs, and the fourth study found that confounding factors may have caused the lung cancers found. Accordingly, the court determined that the studies were of "*no help*." *Id.* at 145-146. The court conceded that trained experts may extrapolate from existing data, but "nothing in either *Daubert* or the Federal Rules of Evidence requires a district court to admit opinion evidence which is connected to existing data only by the *ipse dixit* of the expert. A court may conclude that there is simply too great an analytical gap between the data and the opinion proffered." *Joiner*, 522 U.S. at 146. As in *Joiner*, the only link between exposure to friction products and Plaintiffs' alleged injuries is the proposed *ipse dixit* of Dr. Hammar..

Even more devastating to Plaintiffs' proposed evidence than the absence of *confirming* epidemiological studies is the existence of multiple epidemiological studies indicating a *contrary* conclusion. *Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 882 (10th Cir. 2005) (holding that epidemiological studies are not required to prove causation, but that a substantial body of

epidemiological evidence challenging causation cannot be ignored); *Allen v. Pa. Eng'g Corp.*, 102 F.3d 194, 197 (5th Cir. 1996) (finding it significant that “numerous reputable epidemiological studies covering in total thousands of workers” indicated that there was no causation). Courts have consistently refused to admit expert testimony that flies in the face of contrary epidemiological studies. The Eleventh Circuit Court of Appeals has held that animal research and case reports were properly excluded in the face of overwhelming contrary epidemiological evidence. *Allison, supra*, 184 F.3d at 1316. Likewise, in *Richardson v Richardson-Merrell, Inc.*, 857 F.2d 823 (D.C. Cir. 1988), the District of Columbia Circuit Court rejected proposed expert testimony on the basis of the wealth of epidemiological data to the contrary; *see also Ealy v Richardson-Merrell, Inc.*, 897 F.2d 1159 (D.C. Cir. 1990) (explaining that epidemiological studies were the “measuring stick for the admissibility of expert testimony” on the question whether the drug Bendectin caused birth defects).

Here, there is not only have a lack of epidemiological evidence supporting Dr. Hammar’s conclusory assumption that exposure to automotive friction products causes mesothelioma, but also numerous epidemiological studies showing exactly the opposite. Dr. Hammar rejected these studies because they went against his pre-conceived belief that such a risk should exist. Dr. Hammar instead renders his conclusory opinion presumably on case studies and regulatory actions. However, another court has held: “As support for his opinion, Dr. Hammar relied on various studies and regulatory analyses. The regulatory standards are not probative of scientific analysis or acceptance in the scientific community. The epidemiological studies and meta-analyses do not analyze cases of exposures at very low levels.” *Free v. Ametec, Inc., et al.*, No. 07-2-04091-9SEA (Superior Court of the State of Washington in and for the County of King), Ex. 15.

Therefore, Dr. Hammar's specific causation conclusion, and general causation assumption, is unsupported by relevant science, and should not be admissible under Daubert. *Bitler v. A.O. Smith Corp.*, 400 F.3d 1227, 1233 (10th Cir. 2004) ("When the conclusion simply does not follow from the data, a district court is free to determine that an impermissible analytical gap exists between premises and conclusion."); *Ingram v. Solkatronic Chem., Inc.*, No. 04-CV-0287, 2005 U.S. Dist. LEXIS 38304, at *30 (N.D. Ok. Dec. 28, 2005) ("While the primary concern of this Court is not with the accuracy of [the expert's] conclusions, the Supreme Court has made clear that an expert's conclusions may be the subject of particular scrutiny when they move beyond the boundaries of rational inference into the realm of speculation."). Dr. Hammar's conclusion is unsupported, and in fact is contradicted, by the scientific evidence in the relevant field, and Dr. Hammar posits no alternative proof of but for causation linking mesothelioma to automotive friction products. Without proof, or even a valid assumption, of general causation, there cannot be specific causation, and therefore Dr. Hammar's methodology and conclusions should be inadmissible under *Daubert*.

IV. CONCLUSION

Because Dr. Hammar's opinions are unreliable and do not meet the standard for proper expert causation testimony, Dr. Hammar should be precluded from offering his "every exposure" opinion at trial.

DATED this 7th day of August, 2012.

Dorsey & Whitney, LLP

By /s/ Dan R. Larsen

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CERTIFICATION OF SERVICE

The undersigned certifies that on this 7th day of August, 2012, the foregoing was served on all counsel of record via the electronic service-ECF system.

/s/ Dan R. Larsen